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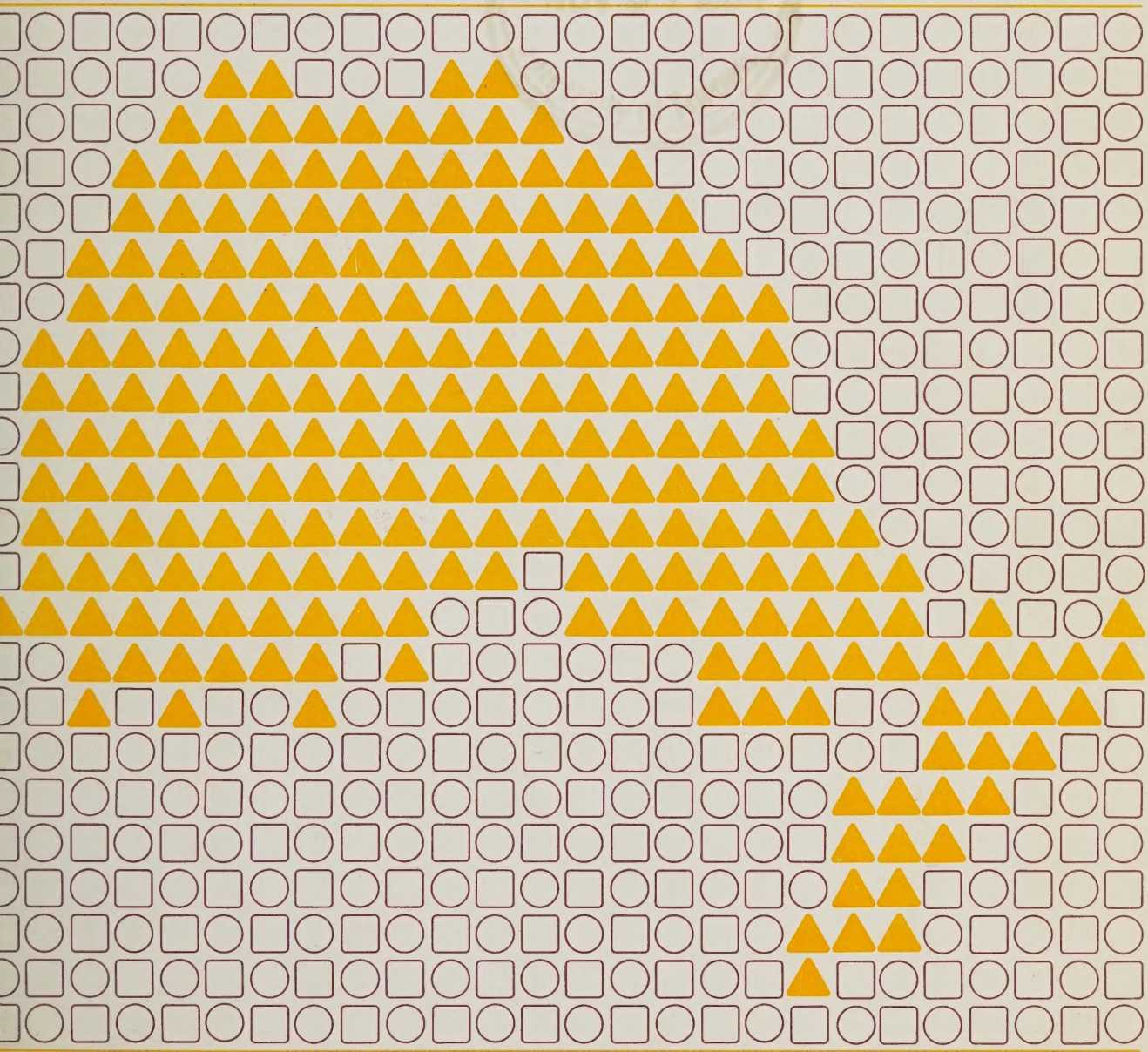
Volume 5, Number 4, October 1984

Hand-Arm Vibration Syndrome

The Medonte Derailment

Automotive Body Repair Shop Hazards

Guideline for Examining Physicians on Surveillance of Silica-Exposed Workers



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Occupational Health in Ontario

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P.L. Pelmear, M.D., F.F.O.M., C.C.B.O.M.

Hand-Arm Vibration Syndrome

Summary

The symptoms, signs and pathophysiology of the Hand-Arm Vibration Syndrome are reviewed. The useful clinical diagnostic procedures are enumerated, together with the steps to be taken to prevent and minimize the ill-effects of the syndrome.

P.L. Pelmear, M.D., F.F.O.M., C.C.B.O.M.,
Occupational Health Branch,
Ontario Ministry of Labour,
400 University Avenue,
Toronto, Ontario
M7A 1T7

Raynaud's Phenomenon, so-named after Maurice Raynaud (1834-1881), may be defined as intermittent constriction of the peripheral vessels - arterioles and veins - with consequent colour change of the skin of the extremities, such as pallor, cyanosis, or both. The phenomenon, commonly precipitated by exposure to cold, may occur primarily, as in Raynaud's Disease, or in association with a number of conditions or diseases.

The primary condition is known as Raynaud's Disease, or Constitutional White Finger. The distinguishing features are the familial predisposition (up to 10 per cent incidence in the population); higher incidence in females than in males (5/1); early onset, with over 60 per cent of those predisposed experiencing the phenomenon by the age of 30; bilateral appearance of the phenomenon in response to stress as well as to cold; and the absence of any predisposing disease or trauma.

The more important secondary causes of Raynaud's Phenomenon include the connective tissue diseases - scleroderma, lupus erythematosis and rheumatoid arthritis; any disease or condition causing proximal obstruction of blood vessels - thoracic outlet syndrome, cervical rib, atherosclerosis, dysglobulinaemia; neurological conditions with muscle impairment - poliomyelitis, syringomyelia; Vinyl Chloride intoxication; injury to the extremities arising from lacerations or frostbite causing immediate blood flow interference; and accumulative trauma, as with vibration, when the vascular changes occur gradually. This state has been termed Vibration White Finger (VWF).

Blanching of the fingers occurs when the arterial muscle spasm, induced by cold, overcomes the intraluminal pressure within the blood vessel.

These various conditions associated with Raynaud's Phenomenon can be distinguished by means of a careful history and clinical examination followed by X-rays and laboratory tests as and when indicated. In field surveys the differentiation is made from the history and clinical examination, but compensation cases and

those referred for treatment require a full laboratory investigation to establish the diagnosis beyond doubt.

Vibration as a cause of Raynaud's Phenomenon was first reported by Loriga in 1911.[1] Compressed air tools were the cause, and this was confirmed by other authors in 1918[2,3] who reported on stonecutters using pneumatic hammers in the limestone quarries of Indiana, U.S.A.

Dr. Hamilton examined 181 stonecutters and carvers, and her description[3] of the symptoms and signs has not been surpassed in the literature since. Her description of the complaints was restricted to the hands. Dr. Cottingham,[2] who examined seven severe cases, gave the symptoms as swelling, vasomotor change and loss of sensibility in the hand, pain, loss of sleep and general nervousness. In his opinion, none of the cases could revert to normal, and he believed that everyone using the hammer must suffer ill effects to some extent.

The U.S. Bureau of Labour sponsored Dr. Hamilton's study because of the concerns raised by unions and Dr. Cottingham. Dr. Cottingham had examined his cases at the request of the union; and the U.S. Bureau, in its final report, noted that Dr. Cottingham's statement was more emphatic than those of the other doctors reporting on behalf of the unions and employers. The bureau report stated, furthermore, that this is perhaps to be explained by the fact that the examinations were made at a time when the use of the air hammer was the subject of sharp controversy between the union and the employers, and the statements made to the doctor by these men — statements that he has accepted unquestioningly — may have been coloured too highly because of the state of war that existed.

With the notable exception of Dr. Cottingham's findings, the symptoms and signs complained of by the workers were all restricted to the hands and arms from this source of localized hand-transmitted vibration. This has been confirmed by numerous researchers since. *Whole body vibration* transmitted to the entire human body through some supporting structure, such as a vehicle seat or a building floor, may give rise to generalized symptoms. They include nausea, epigastric discomfort, loss of weight, reduced visual acuity, insomnia, labyrinthine disorders and muscular and joint pains, but they have been less well documented and evaluated than symptoms and signs caused by hand-arm vibration.

The recognized hazardous work tools and situations for hand-arm vibration exposure now include

compressed air tools used for fettling, hand-grinding and polishing, as well as work involving pedestal grinding, swaging and chain saw operating.

Symptoms and Signs

Following exposure to vibration, the first symptoms are tingling and numbness of the fingers when at rest, particularly at night. Swelling of the fingers, especially over the knuckles, is often noticed as well. Subsequently, after a variable period of time, depending on the dose of vibration received and the sensitivity of the subject, blanching of a fingertip occurs following exposure to cold. The affected finger is numb when blanched and, in severe cases, there may be some persistent numbness or reduced sensitivity between attacks.

With continued exposure to vibration, the blanching attacks become more frequent. The blanching attacks are precipitated by exposure to cold, damp conditions, particularly in the morning and at night, when the subject's metabolic activity is low with a reduced central body temperature. Blanching following the handling of cold objects or immersion in water is a common story. Attacks are more common in winter than in summer, but, eventually, they will occur all year round. Typically, they last from a few minutes to one hour, and terminate with reactive hyperaemia and, often, considerable pain. During an attack, touch, pain and temperature sensitivity are often greatly reduced, and, in some subjects, this may persist causing permanent impairment.

Initially, the blanching is localized to the tips of the fingers most exposed to the vibration source, but, eventually, it spreads to involve all fingers as far as the metacarpo-phalangeal joints and the tips of the thumbs. The palms of the hands are rarely affected. The blanching does not usually occur at work, except during rest periods, but in some subjects the vibration stimulus itself will induce blanching if the fingers are cold.

These symptoms and signs are in response to pathological and physiological changes in the tissues of the fingers. Kadlec and Pelnar,[4] in 1945, on the basis of their plethysmographic studies, disagreed with the then accepted view that VWF was solely a disease of the capillaries,[5] and provided evidence of the crucial role of the finger arteries. They attributed hyperactivity to the muscular layer of the arterial wall, the hypertrophy of which was later documented by Ashe[6] from biopsy and

autopsy specimens. They described two stages of VWF — the first, reversible and, presumably, due to exaggerated spasm and its failure to resolve into vasodilatation under cold; the second, an irreversible stage, demonstrated plethysmographically to be a permanent anatomical obstruction of the fingers' arteries.

The development process is not entirely clear, and several hypotheses have been proposed. It is suggested that the first response to vibration is a relaxation of the small blood vessels and an increase in permeability of the vessel wall with a resulting accumulation of exudate. Hence, the oedema and nerve-end irritation expressed as tingling. Later, hypertrophy of the muscle layer of the blood vessels occurs, with consequent hypersusceptibility to contraction when stimulated by cold. Eventually, the lumen of the blood vessel is reduced due to the muscle hypertrophy and from secondary thrombosis consequent upon a sluggish blood flow. In advanced cases, fibrotic changes occur within the hypertrophic muscle, and this may account for the prolonged spasm with delayed relaxation on re-exposure to warmth, which is a typical feature in such cases, as stressed by Magos *et al.*[7] These pathological and physiological findings have been noted by several authors.[6,8,9]

Ultimately, with complete obliteration of blood vessels, trophic or gangrenous changes may occur, and a few cases have been reported.[9,10,11,12] There are many reports of cyst formation in bones,[13,14,15] but this is a common finding in manual workers. In the subjects studied by James *et al*[16] and Harkonen *et al*[17] there was no statistical difference between the vibration-exposed and manual worker controls. Bone cysts occur because of synovial fluid extrusion from joints and not from reduced blood flow.

The symptoms and signs and the frequency of attacks may be such that, eventually, subjects, apart from taking immediate preventive action to keep warm and avoid cold exposure, have to curtail their activities. Socially, this may mean avoiding outdoor pursuits such as gardening, fishing, swimming and watching outdoor entertainment, and, ultimately, it may involve a change of work to avoid further vibration exposure.

A grading index by stage of symptoms and social/work interference has been described (Table — Stages of VWF) by Taylor and Pelmear,[18] and has proved useful. It was developed following Dr. Taylor's initial investigation of chain saw users in 1969 to express clinically the stages of severity of VWF, and

has since been generally adopted by researchers in the Western World, i.e. North America, Scandinavia, Switzerland and the U.K.

Stages of VWF

Stage	Condition of Digits	Work and Social Interference
0	No blanching of digits.	No complaints.
0_T or 0_N or 0_{NT}	Intermittent tingling, numbness, or both	No interference with activities.
1	Blanching of one or more finger tips with or without tingling and numbness.	No interference with activities.
2	Blanching of one or more fingers with numbness. Usually confined to winter.	Slight interference with home and social activities. No interference at work.
3	Extensive blanching. Frequent episodes, summer as well as winter.	Definite interference at work, at home and with social activities. Restriction of hobbies.
4	Extensive blanching. Most fingers; frequent episodes, summer and winter.	Occupation changed to avoid further vibration exposure because of severity of symptoms and signs.

If the subject is young, with VWF severity no greater than Stage 2, he may well completely recover if he avoids further vibration exposure. If he does not, he will progressively deteriorate. Older subjects and those with severity Grade 3 on discontinuing vibration exposure tend to remain the same, although some continue to deteriorate. Surgery and drug therapy are not generally effective, so prevention is very important.

The prevalence of VWF in working groups, as determined from epidemiological studies, can vary considerably because of labour turnover. Therefore it is not a reliable indicator of the exposure risk. The latent interval, which is the period of time between first exposure to hand-arm vibration and onset of first blanching of a fingertip to cold, is a better indicator for determining the risk factor of a particular work tool or work situation. The latent intervals vary with each situation, but, in general, pedestal grinding varies between three months and 20 years, depending on the

nature of the grinding wheel and the size of the metal piece; fettling varies between four and seven years; swaging averages six months to one year; hand grinding averages five to 15 years; chain saws, three to five years, and anti-vibration chain saws over seven years.

Pathophysiology

In 1862 Raynaud described episodic digital ischaemia in response to cold exposure, and suggested that it was due to sympathetic nervous system hyperactivity. While Lewis,[19] in 1929, attributed the condition of vasospasm, which occurs when the arterial muscle tone overcomes the intraluminal blood pressure, to a local fault within the digital arteries themselves.

In animal studies vasodilatation following sympathetic activity has been shown to consist of passive and active components, the passive being simple withdrawal of sympathetic tone, and the active, a local release of histamine chemical from mast cells around the microvasculature. An increase in sympathetic activity arising from the hypothalamus (coarse control) causes vasoconstriction through noradrenalin stimulation of the muscle fibres in the blood vessels. It also stimulates the mast cells to release histamine, which will cause vasodilatation (fine control) after a delay of seconds. An axon skin reflex is also thought to be involved in stimulation of the mast cells.

Local temperature changes will produce distant effects on the blood flow of the opposite hand via the hypothalamus and the sympathetic system in normal patients. This effect is enhanced in patients with Raynaud's Phenomenon, but the rapid 'post-sympathetic' vasodilatation is delayed or absent.

Lafferty *et al*[20] hypothesise that *Raynaud's Phenomenon* occurs in the pure vasospastic (rather than obstructive) form as a result of a 'local fault' in the histamine release from mast cells. Local trauma, such as vibration or frostbite, could conceivably damage this sympathetic/histamine system by disrupting the mast cells.

Vasoconstriction in the contralateral hand is well recognized and easily demonstrated in cold provocation tests in patients with Constitutional White Finger or Raynaud's Disease. It is less evident in cases of VWF, but may occur to a limited extent. Pyykko *et al*,[21] noticing a similar effect from vibration provocation tests, suggests that this may be due to repetitive vibrations activating the sympathetic reflex, but the more likely explanation is that the blood vessel muscle hypertrophy in the contralateral hand is more

susceptible to stimuli. Exposure to hand-arm vibration is usually bilateral, and muscle hypertrophy has been demonstrated by researchers[8,9] in the blood vessels exposed to vibration.

Numbness of the fingers occurs in vibration-exposed subjects to a significantly greater extent than in workers not exposed. This has been recognized and reported by many researchers since the 1930s. The loss in tactile sensitivity is thought to be due almost entirely to fatigue of the sensory receptors in the skin.

Peripheral neuropathy from vibration exposure has been confirmed by measuring the conduction velocities in the peripheral nerves.[22,23] Lukas,[24] who noted reduced conduction velocities in the terminal parts of the ulnar and median nerves in workers following long vibration exposure times, usually more than 10 years, suggests that the vascular component may play an important role in the development of damage and may be the cause of the neuropathy. But, as observed by Pyykko[21] and others, neuropathy in forest workers bears no relation to the severity of the vascular component, and they may occur independently; so this suggests separate mechanisms.

Muscle fatigue in the hands and arms of forest workers was noted by researchers in the late 1920s,[21] and has been demonstrated objectively by others.[25,26] As the use of vibrating tools is associated with a significant decrease in manipulative dexterity,[27] it seems that the muscular weakness is, at best, partly due to a disturbance of the fine control of the hand muscles. The effect of vibration on muscle tissue and its contraction is not well understood as yet. The reaction of denervated muscle tissue to vibration is to relax,[21] indicating that vibration may directly interfere with the contractile substances in muscle, myosin and actin, preventing their cross-linkage and muscle contraction. In an intact muscle, a momentary decrease in muscle force is also observed when the muscle or tendon is exposed to vibration.[26] Very often, the same subjects with extensive symptoms of neuropathy also have decreased muscle strength, so it seems that degeneration in the nerves that innervate the muscles could be the immediate cause of excessive muscle fatigue.

Clinical Diagnostic Procedure

To make a satisfactory clinical diagnosis, the following procedure should be adopted:

(a) Obtain a complete history of presenting complaints, past medical history, family history

and occupational history with emphasis on nature and length of exposure to vibratory tools.

The history should specifically elicit symptoms of possible collagen or rheumatoid disease, large vessel obstruction, exposure to digital trauma, haemotological dyscrasias and use of vasoconstrictor medications.

(b) Conduct a complete physical examination with emphasis on the musculo-skeletal, cardiovascular and neurological systems to note, particularly, pulses, rashes, skin thickening, trophic changes, joint abnormalities and muscular and neurological defects.

(c) Record response to specific screening tests.

(I) For vascular assessments:
- Lewis Prusik test (nail bed compression).
- Allen's test (wrist vessel compression).

(II) For thoracic outlet assessment:
- Adson's test (rotation of neck on deep inspiration).

(III) For carpal tunnel syndrome assessment:
- Tinel's test (percussion of carpal tunnel).
- Phalen's test (flexion of wrist).

(IV) For nerve impairment:
- dynamometer test (grip strength).
- aesthesiometer tests (depth-sense and 2-point perception).

(d) Routine laboratory investigations.

(I) Blood tests — blood count and differential, ESR, uric acid, rheumatoid factor, antinuclear antibodies, cryoglobulins and serum protein electrophoresis.

(II) Urine tests — urinalysis for proteinurea, and casts.

(III) X-rays — cervical spine and hands to identify cervical ribs, arthritis and cystic changes.

(e) Special investigations as and when indicated.

(I) Cold provocation tests with or without plethysmography and Doppler assessment.

(II) Venography and/or venous radionucleotide scan.

(III) Microvascular assessment with tritiated thymidine.

(IV) Nerve conduction and electromyography.

(V) Vibro-tactile sensitivity measurements.

Assessment

The symptoms, signs and objective evidence accumulated from the above diagnostic procedures can establish with reasonable certainty the well-defined syndrome of Raynaud's Phenomenon and its attributability to hand-arm vibration exposure, if this is the case.

Medical authorities in Europe and America have been using this diagnostic approach in epidemiological studies and for follow-up investigations. The different causes of Raynaud's Phenomenon have been differentiated, together with their related signs and symptoms, and, as mentioned earlier, generalized symptoms or systemic disease has not been found in cases of Hand-Arm Vibration Syndrome. Apart from hand-arm symptoms and signs, the only additional effect in those cases, which has recently been reported by Pyykko *et al*[28] and Iki *et al*[29] has been a susceptibility to increased hearing loss. This has yet to be confirmed by other researchers.

In March 1983 the Scientific Committee on Health Effects of Physical Environmental Factors of the Permanent Commission and International Association on Occupational Health convened a meeting in London, U.K., to discuss the reported existence of 'vegetative' or 'generalized' symptoms in advanced cases described in the Soviet and Japanese literature, and to resolve the divergence of opinion on two important aspects 1) the assessment of the disability in terms of 'stage' classifications, and 2) to what extent segmental vibration *per se* may induce psychosomatic reactions or other effects outside the hand-arm system.

The editors of the published proceedings,[30] Drs. Taylor and Gemne, concluded that the generally

accepted opinion — based on a critical review of the available literature and on physiological and epidemiological considerations — is that there are, at present, no data that confirm the hypothesis that hand-arm vibration causes damage to the autonomic centres in the brain. That the lack of evidence is felt also in Japan was clear from the answers to the questions posed to the Japanese participants concerning these matters.

There was agreement on the overall definition of the *Hand-Arm Vibration Syndrome*. As to disease entity, it is made up of the following components, all peripheral: *circulatory disturbances* ('vasospasm' with local finger blanching, 'white finger'), *sensory and motor disturbances* (numbness, loss of hand and finger dexterity, etc.) and *musculo-skeletal disturbances* (muscle, bone, and joint disorders). The reported influence of hand-arm vibration on endocrine, cardiac, vestibular and cochlea functions cannot be considered established as yet. Further research is considered to be necessary in this field.

Treatment

The use of anti-spastic drugs to reduce muscle tone and spasm in blood vessels has provided relief from attacks of Raynaud's Phenomenon in some patients. Vasodilator drugs, which cause relaxation of the muscle coat of normal blood vessels and subsequent dilatation, have been prescribed for some patients as they were presumed to be helpful. However, all the evidence suggests that the contraction in the arterial wall is too strong in Raynaud's Phenomenon for the drugs to overcome, and unless the drug is given in doses that produce severe side effects, treatment is ineffective.

New therapies continue to be proposed and evaluated, and although most appear to be useful initially, the beneficial effect is not usually maintained. Sympathectomy does not give consistently good results and therefore is less frequently used today than previously.

During an attack of VWF, warm water exposure to the hands while keeping the body cool has been recommended by some, but swinging the arms at the onset of an attack with warmth to the body is probably more useful than any pharmacologic therapy.

For the prevention of attacks, the most useful and important aid is the wearing of warm, dry clothes to maintain a raised body temperature.

Prevention

The important steps to be taken in prevention are:

(a) Identification of hazard by vibration measurement.
(b) Complete avoidance of exposure (if possible) by use of alternative work methods.
(c) Use of anti-vibration pads on tools and chain saws.
(d) Regular and effective maintenance of tools and chain saws.
(e) Education and training of workers to grip tools and chainsaws lightly.
(f) Work rotation to limit exposure.
(g) Medical surveillance, namely, pre-employment and periodic.
(h) Health education of workers to avoid smoking and to wear warm clothes, to maintain a high body temperature to reduce blanching attacks.
(i) Transfer of subjects severely affected.

Only a few follow-up studies of workers using modified chain saws have been reported. Riddel and Taylor[31] noted improvements in men continuing to operate saws with vibration-isolated handles (A/V saws) who were known to have suffered from VWF following the use of saws without vibration-isolation systems. However, some men continued to deteriorate. Three men developed VWF from the use of A/V saws only, but there were compounding factors from previously suspect circulation and motorcycle riding.

In Ontario, Canada, the Workers' Compensation Board (WCB) has received claims for VWF since 1944 and has permitted claims since the early 1950s.

Résumé

Les symptômes, signes et physiopathologie du syndrome de vibration main-bras sont étudiés ci-après. Les méthodes utiles de diagnostic clinique sont énumérées, ainsi que les étapes à suivre pour prévenir et minimiser les effets nuisibles du syndrome.

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Jaan O. Roos, M.D., F.R.C.P.(C)

The Medonte Derailment: A Survey for Health Effects in Firefighters

Summary

Three months after a derailment fire in Medonte Township, Ontario, 104 rescue personnel participated in a survey to determine whether residual health effects, especially respiratory, had resulted from the disaster. Twelve underwent a second examination, and six showed signs that needed follow-up. Several confounding factors made any final conclusion on causality premature. Results do suggest, however, that derailment accidents would be best served by on-site monitoring of acute health effects, and a questionnaire survey of affected participants after the event. Further medical intervention should be recommended by personal physicians.

Jaan O. Roos, M.D., F.R.C.P. (C)., Senior Medical Consultant, Medical Service Chest Clinic, Occupational Health Branch, Ontario Ministry of Labour, 880 Bay St., 2nd Floor, Toronto, Ontario M7A 2C7

The Derailment

A freight train ran off the rails on the morning of Sunday, February 28, 1982, at 8:17 a.m., in a wooded, isolated area near Moonstone, Ontario, and 17 of its boxcars, some loaded with chemicals, caught fire after untracking.[1] While the Medonte Volunteer Fire Department started putting out the flames of the burning boxcars, the freight was identified from the manifest.[2] It included one car containing 39 tons of hydrofluoric acid (HF), a full hopper car of arsenic, cars with drums of butane and hair spray, 30 tons of naptha and isopropanol, hydrochloric acid and resin solution as well as petroleum lubricating oil, steel wool, vinyl acetate, phosphate, skelp, cans of foods and drugstore supplies. The presence of polyvinyl chloride was initially suspected and later confirmed. The HF car, identified as potentially the most dangerous, was not found until the end of the second day. It lay at the bottom of the wreck, but had not leaked. Evacuation orders were set for a five-mile radius, and this area was finally cleared on March 9, ten days after the derailment. In the interval, some 350 volunteer firefighters, mostly farm workers, put out the fire in bitterly cold temperatures, which ranged down to -30°C. They were exposed to dense black smoke; occasionally to severe heat at the fireline, and sometimes to unidentified skin irritant liquid droplet sprays. Many firefighters worked throughout the week despite extreme fatigue.

Public Concerns

In the immediate aftermath of the fire, the roles and responsibilities of public health officials, fire officers, police and railway authorities were debated with vigour.[3] Somewhat later, concern arose about delayed injuries that might have resulted from the prolonged exposure to adverse conditions. While fumes from the HF car were not released, firefighters had worked for long hours under extreme physical distress, and several complained later of various residual symptoms. The community

concern prompted the local fire captain and the medical officer of health to request the Ministry of Labour for medical screening to uncover any resultant illness. The Ministry of Labour was asked to respond when it was learned that a survey team that had previously studied the aftermath of a derailment in Mississauga, Ontario, could be made available for Medonte.

Major derailments involving noxious chemicals had occurred recently near New Orleans[4] and in Mississauga.[5] In these, rescue personnel had been exposed to chlorine gas and there had also been a hazard to a large number of people in adjacent urban areas. Many workers had been exposed for several days to sublethal doses of irritant gases, and several studies were underway to determine any residual injuries to the respiratory system. This background, augmented by media attention, led to a general awareness of the hazards, including perceived insidious late effects, faced by firefighters, police and clean-up work personnel.

Case reports on the late effects of fires or irritant fumes, were, however, reassuring.[6,7,8] The New Orleans study, which had revealed several early deaths due to chlorine inhalation, and the local Mississauga survey, which dealt with a derailment involving less severe exposures, had suggested that late respiratory sequelae did not occur unless early problems were very marked.

Extent of the Problem

Four major hazards were present in the Medonte derailment: extreme cold from the February weather, extreme heat from the fireline, fatigue from ten days of hard physical work and the risk of trauma from slips on the water-frozen, steep embankments of the track. In addition, there was respiratory tract and skin exposure to fumes and particulates from the fire as well as to spray from irritant liquids. About 350 persons were involved with the fire and clean-up operations.

Survey Methods

The call for a survey was made about two months after the fire, but because of difficulties in notifying the subjects, the survey could not be carried out until May 27, 1982, when all persons known to have been involved with rescue operations at the wreck were invited to a medical screening at the Moonstone fire hall. Of the approximately 350 individuals contacted, 104 presented for the survey. All but two were male and all were Caucasian.

A chest physician interviewed all by questionnaire and performed a physical examination of the respiratory system as well as other relevant systems as indicated by answers to the questionnaire. A pulmonary function technologist supervised forced expiratory spirometry. The questionnaire (Ross C. Kory, Respiratory Questionnaire (unpublished)) emphasized symptoms expected in firefighters and also reported smoking history, past health problems and details of exposures at the fire site. Forced expiratory spirometry was done with a portable dry wedge spirometer (Vitalograph Model S™) capable of measuring volume up to 7.5 L over a time of 12 seconds. The instrument was checked for calibration with a three-litre syringe before, during, and after completion of the survey. At least three acceptable tracings were performed according to criteria recommended by the American Thoracic Society[9] and the reference values used were those of Crapo, et al.[10]

Seventeen persons who complained of symptoms or who showed signs conceivably associated with the fire were invited back for a follow-up survey on September 14, 1982, by the same examiners, using the same techniques, and 12 were able to return for the re-examination. Where abnormal findings persisted, subjects were advised to seek further treatment and follow-up advice from their personal physicians. Interim and re-examination reports were sent with the subjects' permission to their personal physicians together with a preliminary summary of findings in the group. All results were also reported to the referring medical officer of health.

A comparison of spirometric findings was made with groups of firefighters from Etobicoke, Ontario, and the neighbouring community of Mississauga, who had recently been studied by the same team. The Etobicoke men had not had unusual exposure to respiratory hazards beyond that encountered through their regular full-time firefighting duties, whereas the Mississauga group had recently worked on a major derailment involving exposure to low levels of chlorine and other irritant gases. No persistent difference had been found between these two groups, but both differed from the Medonte subjects in that the Toronto area men were full-time firefighters and the Medonte men were all volunteers with primary occupations mostly in farming. Other differences were that the Mississauga fire had been allowed to burn out by itself while the firefighters remained, for the most part, at a distance and the essential hazard was a pervading low level of chlorine

gas. The Medonte men worked considerably closer to the fire, and the respiratory hazards were probably higher levels of combustion products and heat.

Results

Table 1 summarizes the anthropometric information. In general, the Medonte firefighters were very similar in mean age and height to those from each of the other two studies. The average spirometric values for the Medonte firefighters are shown in Table 2; they were near the predicted means for the average age and height of the group, and were also similar to the values for each of the two other groups of firefighters.

Table 1

Anthropometric Data (± 1 S.D.)

	Height (cm)	Age (years)
MEDONTE	177.8 (± 5.1)	35.7 (± 12.1)
Mississauga	178.3 (± 4.2)	35.9 (± 8.3)
Etobicoke	178.0 (± 4.5)	34.9 (± 8.3)

Table 2

Spirometry

	FVC (±1 S.D.) L	FEV1 (±1 S.D.) L	FEV1/FVC %
MEDONTE	5.18 (± 0.64)	4.07 (± 0.66)	78.6
% of predicted[10]	98	95	
Mississauga	5.35	4.30	80.4
% of predicted[10]	102	100	
Etobicoke	5.60	4.05	72.3
% of predicted[10]	106	94	

Thirty-eight Medonte firefighters had never smoked; 12 had quit smoking, and 54 were currently smoking. The amount of cigarette smoking ranged from three to 75 cigarettes daily; the majority averaged 20 to 25 per day. A few smoked very heavily and their consumption is shown in Table 3.

Sixteen persons were entirely free of symptoms, had normal spirometry and had no abnormality on chest examination. Of those, 10 had never smoked, three had quit and three were smokers, two of the latter smoking very heavily. The mean age of the asymptomatic individuals was 36 years, and, thus, did not differ from

the mean age of the entire group. The smoking histories are summarized for the symptom-free group in Table 4.

Table 3 **Smoking Histories**

Never smoked	38 firefighters
Ex-smokers	12
Pipe/cigar smokers	9
1-15 cigarettes daily	13
20-25 cigarettes daily	23
35-40 cigarettes daily	4
50 cigarettes daily	4
75 cigarettes daily	1
Total	**104**

Table 4 **Asymptomatic Subjects: Smoking Histories**

	No. of Subjects	Age range (years)
Never smoked	10	22-52
Ex-smokers	3	36-49
15 cigarettes daily	1	31
35-40 daily	1	35
50 daily	1	48

Symptoms or signs, agreed by both examiner and examinee to be unlikely to be related to the derailment episode were found in 71 workers. Their severity ranged from trivial to fairly marked, and they were reported to their family physicians. Thus, 68 per cent of the surveyed population did have symptoms, mainly from the respiratory system, which seemed to be unrelated to the derailment exposure. This is consistent with the known high prevalence of respiratory morbidity in the population as a whole.

Perceived residual effects by the firefighters' own statements were noted in 17 subjects. Five had associated spirometric ventilatory defects of slight to moderate degree, and all were cigarette smokers. Twelve others reported residual symptoms, temporally related to the derailment, but showed no spirometric abnormalities. Of these, three were non-smokers, one smoked only minimally, and eight were current smokers.

The symptoms that were temporally related to the event by 17 firefighters at the time of the initial examination included: non-productive cough and/or

wheezing (six), productive cough (four), peeling of skin of hands (two), excessive nasal mucus production or nasal congestion (three), sore throat (one), change of colour or consistency of sputum (two), exertional dyspnoea (one), and haemoptysis briefly after the fire (one). Crackles at the base of the lung were heard in one subject, and coarse, persistent localized wheezes in two. A slight peeling of skin on the hands was confirmed by inspection in one subject. The spirometric findings and smoking histories are recorded in Table 5.

The 17 firefighters who had abnormalities possibly related to the fire were recalled for another examination 3½ months later on September 14, 1982. Twelve were able to return, and reminder notes were sent to family physicians regarding the remaining five who did not.

Subjects with continuing symptoms at the second examination complained of the following: one man with new cough, sputum, and wheezing while lying down, continued to have diminishing degrees of these symptoms; spirometry remained normal. Another, with nasal mucus production and earlier skin peeling was improving; spirometry was normal. One man, a never-smoker, continued to have cough and wheezing without change since the initial examination; while spirometry remained within normal limits on both occasions, a trivial drop was noted on the second examination; auscultatory wheezes were also present at that time, and asthma was suspected. One moderate smoker had slight airflow limitation on both spirometric tests, which could not be reversed with bronchodilator aerosol; his original complaints of exertional dyspnoea, which had started after the fire, and of wheezing, which had pre-dated the fire, had, however, cleared by the second examination. A moderate smoker with normal physical and spirometric findings reported a non-productive daytime cough, new since the derailment at the first examination; he was found to have diffuse end-expiratory wheezes on the second study. A minimal smoker with higher than average exposure to the fire fumes reported haemoptysis as well as a cough starting after the fire. The cough persisted at the time of the re-examination and, at that time, he had also developed a new fixed wheeze on auscultation. The haemoptysis, however, had not recurred. Thus, of the 12 who were re-examined, six had continuing symptoms or had developed new respiratory system abnormalities in apparent association with the aftermath of the fire. All histories and observations were reported to family physicians, and particular attention to follow-up was recommended.

Table 5	Perceived or Observed Residual Health Effects			
Subj.	First Examination	Second Examination	Spirometry	Smoking
11 •	Productive cough	Decreasing	Normal	25
12 •	Skin rash Nasal mucus	Improving Improving	Normal	25
13	Basal crackles	(No re-examination)	Normal	Pipe/cigar
19 •	Cough and wheeze	Continuing, no change	Normal	Never
32	Nasal congestion Sore throat	Cleared Cleared	Normal	Never
36	Change of sputum colour	Cleared	Normal	25
38	Exertional dyspnoea	Cleared	Slight obstruction	15
39 •	Dry cough	Cough cleared New wheezes heard	Normal	15
48	Wheezing	(No re-examination)	Mod. obstruction	20
53	Wheezing	Continues to wheeze, now states that wheeze pre-existed fire	Slight obstruction	25
55	Wheezing, cough, sputum; had child-hood asthma	(No re-examination)	Normal	10
59 •	Increased cough & sputum; change in sputum colour	Continuing unchanged new wheezes heard	Slight obstruction	25
70	Wheeze heard	Wheeze cleared	Continuing slight obstruction	Quit; (35/d x 14 y)
79 •	Cough, haemoptysis	Cough persisted; haemoptysis cleared	Normal	3
7	Skin rash	Cleared	Normal	Pipe/cigar
64	Nasal mucus; Expiratory wheeze	(No re-examination)	Normal	25
72	'Phlegm from throat' Localized wheeze	(No re-examination)	Normal	Never

• = Special follow-up recommended

The remaining six workers had improved. One had cleared of his initially reported skin rash and slightly productive cough while continuing to have normal spirometry. One had decreasing skin rash and decreasing nasal hypersecretion as well as continued normal spirometry. One, with initial slight nasal congestion and sore throat, had cleared completely by the second examination, and continued to have normal spirometry. Another with pre-derailment cough, wheezing, and white sputum had developed green sputum a month after the fire, but reported no continuing symptoms at the follow-up examination; his spirometry remained normal. A moderate smoker continued to have slight airflow limitation, irreversible with bronchodilator, but his symptoms of wheezing and initial exertional dyspnoea had cleared. One, with slight airflow limitation at the first examination, continued to have fine end-expiratory wheezes, but he felt that these had been present before the fire; he continued to smoke, and associated his symptomatic wheezing with changes in his smoking dose. A fixed, coarse auscultatory wheeze, noted in one man with slight airflow limitation, had cleared, although the obstruction on spirometry remained unchanged. He had also had previous respiratory symptoms, and had, until recently, smoked about 35 cigarettes daily for 14 years. His exposure at the fire site had been above average. In this remaining group it was thought to be unlikely that the fire exposure had had a significant impact on health; any residual symptoms were more probably associated with histories of smoking. Since many of the workers were farmers, it was also of interest to hear that symptoms were aggravated or produced in some instances by wet hay. This introduced an additional confounding factor to the cigarette smoking.

Implications of the Survey

The Medonte survey was undertaken to answer community concerns about the residual effects on the health of emergency personnel. Of about 350 workers at risk, 104 chose to be examined three months after the accident, and 17 were called back for a re-examination at 6½ months because of a suspicion of adverse health effects. Twelve of those presented for the second examination. At that time, six volunteer firefighters continued to show symptoms or signs that suggested further specific follow-up; four of these were smokers of 15 to 20 cigarettes daily, one smoked three cigarettes daily, and one, with definitely continuing symptoms, had never smoked. The remaining six had symptoms or signs that had either disappeared

completely, were definitely improving or they stated at the time of the recall study that their symptoms really existed before the fire. Of this latter group, one had never smoked, one was an ex-smoker with a previously heavy smoking history and the others smoked 15 to 25 cigarettes or pipe or cigars daily.

Both the initially gathered medical findings and the changes noted on the follow-up examination were reported to family physicians and to the medical officer of health.

Conclusions

The short follow-up of this screening survey did not permit conclusions about whether permanent adverse health effects occurred to any of the firefighters studied, although several subjects with new symptoms should benefit from the longer period of observation that was recommended to their family physicians. The usual confounding effects of cigarette smoking and, in some subjects, pre-existing bronchitis or asthma made early conclusions difficult. A further diagnostic problem was posed by the wheezing and coughing that some of the volunteer firefighters associated with exposure to wet hay during the course of their normal farmwork activities. Nevertheless, clinical data starting three months after the derailment are available to provide a comparison for any persistent abnormalities that may become apparent under the follow-up of family physicians.

Conclusions that are possible are that upper and lower respiratory tract irritation and some skin irritation did occur acutely, with clearing within a few weeks in most subjects, but with a slower improvement tendency in a few. Pre-existing symptoms of chronic bronchitis or asthma were common, and, in some subjects with such preceding complaints, sputum or nasal mucus production were transiently increased. In a few, superimposed infection probably occurred, as indicated by the reports of colour change in sputum. Spirometry did not contribute significantly to case-finding, but it helped to reassure firefighters in some instances that no spirometrically measurable loss of lung function had occurred.

These initial findings are consistent with those from other studies, which indicate that late serious health effects are unlikely to occur if the initial acute effects are only slight or moderate.[11-15] The changes that were noted could probably have been satisfactorily recorded on a standard self-administered respiratory

questionnaire, augmented by questions relevant to firefighting and to skin problems. Thus, in future surveys under similar circumstances, rescue personnel would be best served by on-site monitoring of acute effects by medical or para-medical personnel, followed, in the aftermath, by a standard questionnaire, modified to correspond to any known specific health hazards. The answers to the questionnaire would then prompt examinations and follow-up by family physicians as required. A more elaborate centrally directed survey does not appear to have any additional advantage, and with proper explanations, community concerns should also be well answered with this approach. An additional advantage of a questionnaire survey would be that there would be none of the logistic problems of getting workers to a specified survey location, and that all known persons in exposure would have the benefit of a screening questionnaire, most probably earlier than might be possible with a mass examination.

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Résumé

Trois mois après un incendie survenu dans le canton de Medonte (Ontario) à la suite d'un déraillement, 104 secouristes ont participé à une enquête pour déterminer si le désastre leur avait laissé des séquelles, notamment des troubles respiratoires. Douze secouristes ont fait l'objet d'un deuxième examen, et six d'entre eux présentaient des symptômes qui nécessitaient un suivi. Plusieurs facteurs déconcertants ont rendu prématurée toute conclusion quant à la causalité. Les résultats laissent toutefois entendre que les meilleures initiatives à prendre après un déraillement consistent à procéder à une surveillance sur les lieux des effets aigus sur la santé et à faire remplir un questionnaire aux personnes touchées. Les médecins de famille devraient leur recommander une autre consultation médicale.

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J.G. Collinson, P.Eng., C.I.H.
G.S. Rajhans, M.Sc., (Eng.), P.Eng., C.I.H.

Automotive Body Repair Shop Hazards

Summary

Results of industrial hygiene surveys of five autobody repair shops to determine the degree of hazard associated with the use of isocyanate-containing paints and hardeners are reported.

Sampling trains of two midget impingers in series, each containing a nitro-reagent absorbing solution, were used. Analysis was by high-speed liquid chromatography. Results ranged from less than 0.001 ppm to 0.003 ppm. However, the isocyanate compounds in the paints are present in a prepolymerized form, and the combined exposure to the monomeric and polymeric forms may be in excess of 0.005 ppm.

Suggestions are made regarding exhaust ventilation and personal respiratory protection to reduce the spray painter's exposure to a minimum.

J.G. Collinson, P.Eng., C.I.H.,
G.S. Rajhans, M.Sc (Eng.), P.Eng., C.I.H.
Occupational Health Branch, Ontario Ministry of Labour
400 University Ave., Toronto, Ontario M7A 1T7

Automotive Body Repair

The automobile body repair industry has grown at the pace of the automobile manufacturing industry so that, today, thousands of companies in Canada are involved in the repair and refinishing of car exteriors. In the early days, car bodies were mounted on separate ladder chassis and had hardwood frames to which cloth and metal panels were fastened. Since World War II, the majority of mass-produced automobiles have been manufactured with an all-steel body, but the methods used to repair them have not changed significantly.

The basic steps in the repair and refinishing of a damaged or rusted car body are:

- Dented areas of metal work are hammered out to the correct shape using a variety of dolly blocks and hammers. Areas that are too corroded or too severely torn to hammer into shape are cut out and replaced with new sheet metal spot-welded to the existing body.
- Rust is removed with a power disc grinder. A fairly recent innovation has been the use of small compressed air-powered abrasive blasting equipment.
- The remaining imperfections are filled with body putty and the whole area is sanded down to a smooth even contour, either by hand or by power sanding, usually with 320-grit paper.
- The area is then treated with a metal conditioner applied to any bare metal areas and with one or more coats of primer/surfacer, usually a grey or red oxide. When dry, after about 30 minutes, the surface is sanded with 400-grit paper followed by rubbing compound.
- The area is cleaned of all rubbing compound residue and, finally, sprayed with two or three light colour coats of paint, spaced approximately 15 to 20 minutes apart, until full hiding is achieved over the primed areas.
- The vehicle is then left overnight in a dust-free environment. This generally means that it cannot be removed from the spray booth for 12 to 16 hours.

In compressed air spraying, liquid paint is drawn through a nozzle and atomized by a high velocity jet of

air. The flow of air conveys the finely atomized droplets to the object being painted. The stream of air is deflected when it strikes the object. Paint droplets of sufficient mass are not deflected with the airstream, but are deposited on the object. Droplets under 12 micrometres in diameter (approximately) do not have sufficient mass and are deflected with the airstream. This results in 'bounce back' of around 20 per cent of paint droplets, and additional paint is lost when the spray pattern does not completely contact the object.[1]

The compressed air pressure at the spray gun usually ranges from 40 to 60 pounds per square inch.

Occupational Hazards

Solvents

The variety of solvents and reducers used in the industry generally contain a mixture of aromatic and aliphatic hydrocarbons, ketones and esters. While the compounds have different levels of toxicity, they all produce, at sufficiently high concentrations, a narcotic effect with depression of the central nervous system. In addition, aromatic hydrocarbon compounds are absorbed through the intact skin, and, in general, are more irritating and narcotic than aliphatic hydrocarbons. Aliphatic hydrocarbons, such as mineral spirits, are surface irritants, defatting the protective oily layer of the skin, which may lead to dermatitis.

Polyurethane Paints

During recent years, the manufacturers of automotive paints for the refinishing market have developed polyurethane additives, which, when added to alkyd and acrylic enamels, improve the gloss of the finish, improve the resistance of the coating to chipping and shorten the drying time substantially. As a result of these benefits, particularly the decreased drying time, there has been a gradual switch to these products, so that at this time, we believe that the majority of body shops are using polyurethane hardeners in their paints on a regular basis.

The polyurethane hardeners contain, in addition to a variety of solvents, polyisocyanate compounds, usually isophorone diisocyanate (IPDI) and hexamethylene diisocycanate (HDI).

Typically, to repaint a full-sized North American car, four litres of paint are required. To this quantity of paint is added 500 millilitres of hardener, plus two litres of reducer, resulting in 6.5 litres of paint. The hardener

Table 1		Metallic Pigments Found in Automotive Paints	
Metal	**TWAE (mg/m^3)**	**Pigment Colours**	**Remarks**
Antimony	0.5	Orange	
Barium	0.5	Yellow Orange Red	
Cadmium	0.05	Red	Proposed as designated substance in Ontario
Chromium (as CrO_3)	0.05	Yellow Orange	Proposed as designated substance in Ontario
Cobalt	0.05	—	Used in paint drier
Copper	1.0	Blue Green	
Lead	0.15	Yellow Orange	Designated substance in Ontario
Molybdenum	10.0	Orange	
Nickel	0.1	Yellow	
Selenium	0.2	Red	

is packaged in 500-millilitre containers so that a full container of hardener is used with a four-litre container of paint.

Isocyanates are chemical compounds containing the Nitrogen-Carbon-Oxygen group (NCO). They react readily with a wide variety of compounds containing hydrogen atoms to produce polymers called polyurethanes. Polyurethanes are, in turn, used to produce, in addition to paints and coatings, flexible and rigid forms, synthetic rubbers and adhesives. The technology originated in Germany during the late 1930s, and, after World War II, spread worldwide.

The main isocyanate compounds used in the production of the various polyurethanes are toluene diisocyanate (TDI), diphenylmethane diisocyanate (MDI), naphthalene diisocyanate (NDI) and the two diisocyanates previously mentioned, HDI and IPDI, used in the manufacture of polyurethane coatings and paints.

The isocyanate compounds in paints and coatings are generally prepolymerized, i.e. bound or blocked in a reversible reaction with other chemicals so that the free isocyanate is safely held in chemical combination until the bonds are broken by heating or by the chemical displacement of the blocking agent with compounds containing more highly reactive hydrogen atoms. Even under these conditions, a free isocyanate level of 0.5 to 2.0 per cent will exist, depending upon the length of storage.[2]

Paint Pigments

Table I lists a number of metallic pigments commonly found in automotive paints, together with the allowable time-weighted average concentration for occupational exposure (TWAE) in Ontario to each of them. Also indicated are the metals proposed for designation in Ontario under the *Occupational Health and Safety Act.*

Lead chromate is also an example of an insoluble hexavalent chromium compound that is recognized to be carcinogenic in humans. There are a number of reports showing an increased incidence of lung cancer among workers involved in the manufacture of chrome pigments.[3] As a result, the TWAE for certain water insoluble hexavalent chromium compounds, including chrome pigments, is 0.05 milligrams per cubic metre.

The other metallic pigments in Table I are considered to have varying degrees of toxicity, ranging from molybdenum, the insoluble compounds of which are considered to be relatively innocuous, to highly toxic cadmium compounds, with a TWAE of 0.05 milligrams per cubic metre.

Body Filler

Body filler, or putty, consists generally of polyesters and styrene monomer to which a catalyst, usually methyl ethyl ketone peroxide, is added. The TWAE for styrene monomer is 50 ppm and for methyl ethyl ketone monomer the TWAE is 0.2 ppm.

Once the body filler has hardened, usually in about five minutes, the cured polyester resin is considered to be of low toxicity with a TWAE of 10 milligrams per cubic metre for total dust. When the material is ground to the body contour, considerable quantities of dust are produced, which may also contain toxic paint pigments from the existing vehicle finish and, possibly, lead from solder used to fill seams in the body during the car's manufacture.

Abrasive Blasting

Abrasive blasting is being used by an increasing number of companies to remove rust more thoroughly and to obtain an improved metal surface for painting. Since the operation is extremely dusty, it is usually performed outdoors as far away from the shop building as possible.

Silica sand, which is the common abrasive, contains a high concentration of crystalline free silica. Its inhalation can cause silicosis.

Noise and Vibration from Power Tools

Workers are exposed to noise levels well in excess of 90 dBA during the use of compressed air-driven tools, such as chisels, rotary sanders and impact wrenches.

Noise levels, measured at the operator's ear, ranged from 99 dBA while using a small impact wrench to 119 dBA using a power chisel inside the passenger compartment of an automobile. Disc and linear sanders generate noise levels of about 105 dBA.

Vibration measurement of hand tools is now feasible, and acceleration levels in excess of 5 ms^{-2} or 134 dBA should give rise for concern if the worker uses the tool for more than four hours per day. Where necessary, preventive measures as outlined in the most recent draft international standard ISO/DIS 5349 document and the NIOSH Current Intelligence Bulletin, should be instituted.[4,5]

Isocyanates

Isocyanates may present a significant health hazard due to their irritant and sensitizing properties. Inhalation of isocyanate vapours or aerosols such as those generated in paint spraying may result in primary irritation of the respiratory tract, sensitization to isocyanates and a gradual loss of pulmonary function.

Isocyanates have been prescribed as a designated substance in Ontario (Regulation 455/83) and TWAEs have been set for four of the most common isocyanates - TDI, MDI, HDI and IPDI.

The lowest level at which irritation from isocyanate exposure in humans has been reported is 0.03 ppm; however, several studies have shown that sensitized workers will react to TDI vapour at concentrations of 0.001 ppm.[6]

Table 2

Company	Sampling Duration (Minutes)	Weight Collected (Micrograms)		Airborne Concentration (ppm)		Sampling Location
		HDI	IPDI	HDI	IPDI	
A	83	<1.0	<1.0	< 0.002	< 0.001	Side wall, midway
	83	<1.0	<1.0	< 0.002	< 0.001	Exhaust wall, midway between exhaust plenums
	83	1.6	1.5	0.003	0.002	Opposite side wall, inlet end
	83	<1.0	<1.0	< 0.002	< 0.001	Opposite side wall, exhaust end
B	49	<1.0	<1.0	< 0.003	< 0.002	Side wall, midway
	49	<1.0	<1.0	< 0.003	< 0.002	Exhaust wall, midway between exhaust plenums
	49	<1.0	<1.0	< 0.003	< 0.002	Opposite side wall, inlet end
	49	<1.0	1.1	< 0.003	0.002	Opposite side wall, exhaust end
C	49	<1.0	<1.0	< 0.003	< 0.002	Side wall, midway
	49	<1.0	<1.0	< 0.003	< 0.002	Exhaust wall, midway between exhaust plenums
	49	<1.0	<1.0	< 0.003	< 0.002	Opposite side wall, inlet end
	49	<1.0	<1.0	< 0.003	< 0.002	Opposite side wall, exhaust end
D	53	<1.0	<1.0	< 0.003	< 0.002	Side wall, midway
	53	<1.0	<1.0	< 0.003	< 0.002	Exhaust wall, midway between exhaust plenums
	53	<1.0	<1.0	< 0.003	< 0.002	Opposite side wall, inlet end
	53	<1.0	<1.0	< 0.003	< 0.002	Opposite side wall, exhaust end
E	120	<1.0	<1.0	< 0.001	< 0.001	Side wall, inlet end
	120	<1.0	<1.0	< 0.001	< 0.001	Opposite side wall, inlet end
	120	<1.0	<1.0	< 0.001	< 0.001	Side wall, in front of exhaust plenum

< denotes 'less than'

Investigation of Isocyanate Exposure

Since the fall of 1982, the Occupational Health Branch has performed industrial hygiene surveys of five body shops in Northern Ontario following complaints of respiratory illness by workers using polyurethane paints and hardeners. In each case, a walk-through survey was performed, together with sampling for airborne HDI and IPDI during spray painting. The sampling method used was that described in the Code for the Measurement of Airborne Isocyanates (Ontario Regulation 455/83), in which a battery-operated pump is used to draw a known volume of air at a flow rate of one litre per minute through a collection train consisting of two midget impingers in series, each containing a nitro reagent absorbing solution. Sampling trains mounted on tripods were positioned on both sides of, in front of and behind the vehicle to be sprayed, as close to the car as was practical. Sampling was conducted during actual spray painting only, and the pumps were shut off during the 15 to 20 minute 'flash-off' period required between coats. The sampling trains were not mounted directly onto the painter because the impingers were not spill-proof.

All of the body shops used a side-draft spray booth, and in all but one instance, the booths were prefabricated units with an exhaust flow rate of approximately 12,000 cubic feet per minute (cfm). The fifth company had built its own booth, and the exhaust flow rate was only 5,000 cfm. None of the companies had provided any local exhaust ventilation at the paint-mixing bench.

At the Occupational Health Laboratory, the impinger contents were analysed for HDI and IPDI using high-speed liquid chromatography. The minimum detection level for the two compounds with this method is one microgram. The precision of the analytical method is such that for a standard solution having a concentration of one microgram per millilitre, the per cent relative standard deviation is reported to be 5.2 per cent for IPDI and 2.5 per cent for HDI. The sampling results, enumerated in Table 2, reveal that all but two samples contained less than the lower detection limit of one microgram of HDI and IPDI.

One of the exceptional samples, collected at Company 'A' on a side wall of the spray booth near the end through which make-up air was admitted, revealed an HDI concentration of 0.003 ppm and an IPDI concentration of 0.002 ppm. The total monomeric isocyanate concentration in this case was, therefore,

0.005 ppm. The other sample was collected at Company 'B' near the exhaust plenum of the spray booth, and the concentration of IPDI was less than the lower detection limit of 0.003 ppm. The hardener used during these surveys had a total isocyanate content of 14.75 per cent, and the air sampling results confirm that the fraction of free monomeric isocyanate is very low.[7]

The estimation of actual worker exposure based on area sampling is difficult, but from experience gained by sampling for a variety of contaminants with concurrent area and personal samples, it is believed that, generally, worker exposure is under-estimated by area sampling rather than over-estimated.

The time-weighted average exposure of an automotive paint sprayer will, of course, be far lower than the value suggested by the air sampling since the number of hours spent per day does not exceed one to two hours in most cases.

Control Measures

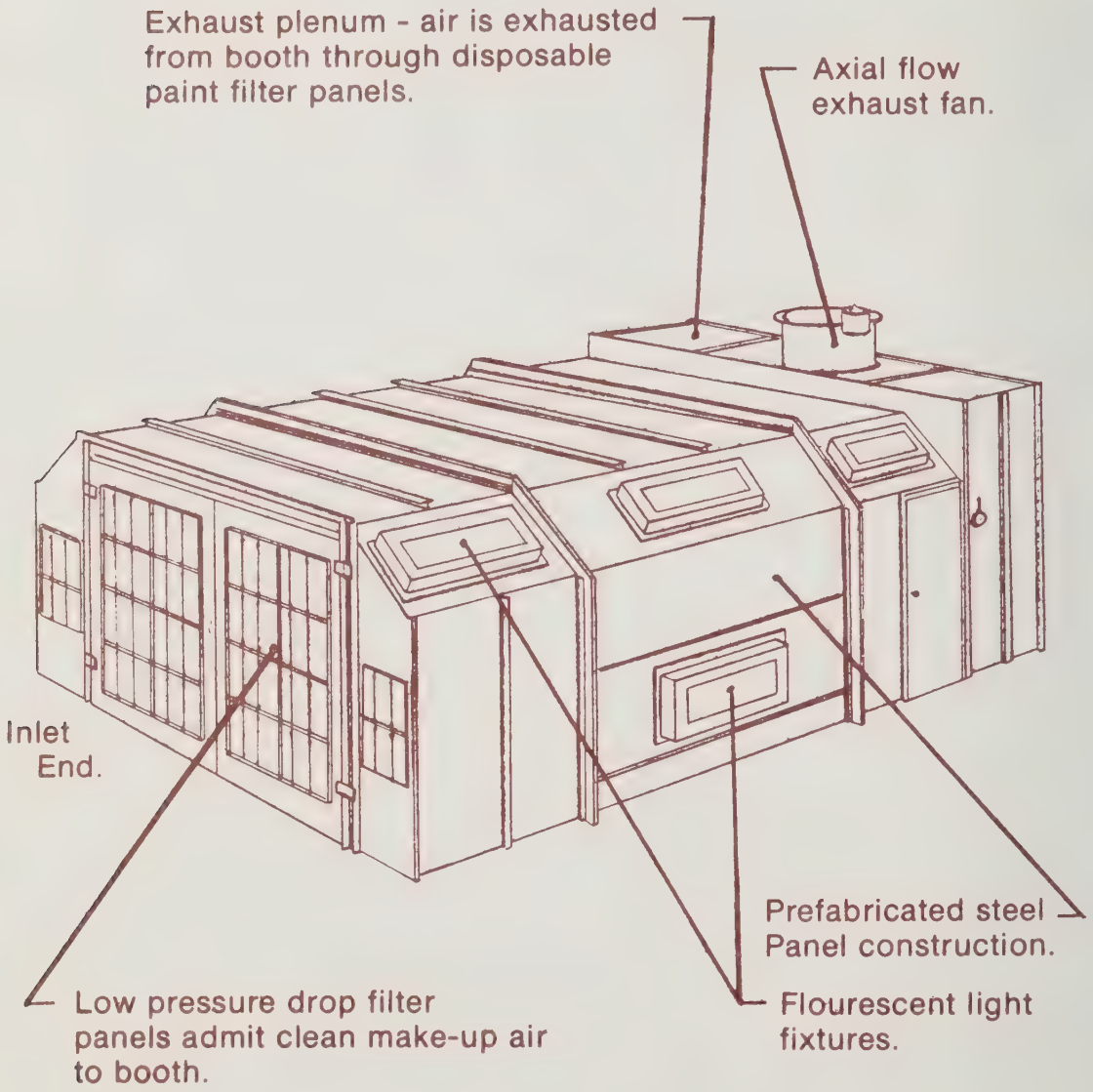
Because of the extremely low concentrations at which sensitization to isocyanates can develop, a combination of local exhaust ventilation and personal respiratory protection is recommended for automotive spray painters using isocyanate-containing paints. Spray booth ventilation effectively isolates isocyanate vapours and prevents exposure of workers in the general shop area, while the respirator effectively protects the spray painter. There are too many variables that can affect the airborne concentration during painting to rely on ventilation alone. Differences in paint viscosity and compressed air pressure of the spray gun may increase the amount of paint deflected into the air due to 'bounce back,' or the painter may need to stand upstream of the exhaust ventilation in order to paint a certain area of the car. A respirator is the only means of assuring the painter the lowest practical exposure.

Spray Booth

The majority of spray booths are prefabricated modular units supplied by one of the specialized companies that cater to the spray coating industry (Figure 1). The nominal dimensions for automotive booths appear to be 24 feet long, 14 feet wide and nine feet high. A large set of doors in one end enables the car to be driven into the booth, and, at the opposite end, there is a full width plenum with a large area of dry filters, from which air is exhausted at a flow rate of approximately 12,000 cfm.

Make-up air from the rest of the shop is admitted to the booth at the opposite end through filter panels in the doors. This arrangement provides air velocity down the empty booth of about 100 feet per minute, the value recommended by the American Conference of Governmental Industrial Hygienists,[8] and the presence of a car in the booth will reduce the cross-sectional area, thus increasing the velocity. The exhaust fan is a belt-driven, axial flow-type, designed to operate at a low static pressure of one-half inch water column.

Figure 1 **A Typical Prefabricated Sideflow Automotive Spray Booth**


Exhaust plenum - air is exhausted from booth through disposable paint filter panels.
Axial flow exhaust fan.
Inlet End.
Prefabricated steel Panel construction.
Low pressure drop filter panels admit clean make-up air to booth.
Flourescent light fixtures.

In another prefabricated automotive booth design, air is introduced through a large area of filter panels in the ceiling and exhausted by way of a floor plenum. This design also calls for an exhaust volume of 12,000 cfm, which results in a lower air velocity than the horizontal flow booth.

Due to the flammability of paints and solvents, the booth should be fabricated from steel and other fire-resistant materials. All lighting and other electrical equipment should conform to the Regulations for Class 1, Group D locations of the Ontario Hydro-Electric Safety Code. The exhaust fan should have non-ferrous blades and be belt-driven by a motor mounted outside of the exhaust duct.[9]

Respiratory Protection

Respiratory protection, worn by spray painters when applying isocyanate-containing paints, must conform to the Code for Respiratory Equipment for Isocyanates (Appendix). The type of respirator required is a positive pressure supplied-air respirator with full facepiece or hood or self-contained breathing apparatus with full facepiece. Obviously, in a spray booth where a supply of compressed air already exists to power the spray gun, the supplied-air respirator is more practical, and is the type used in industry. A respirator should also be worn during the mixing of the hardener into the paint, and, in many body shops, this operation is performed inside the spray booth within reach of the supplied-air respirator.

The compressed breathing air must meet the purity requirements of CSA Standard Z180.1, entitled 'Compressed Breathing Air'. In this standard, the carbon monoxide concentration in the air shall not exceed 20 ppm; the carbon dioxide concentration shall not exceed 1,000 ppm and condensed hydrocarbons shall not exceed 50 milligrams per cubic metre. Therefore, when plant compressed air is used as breathing air, a trap and carbon filter must be installed in the line to remove oil, water, scale, odour and taste.[10] The airflow to the respirator must be not less than four cfm and must not exceed 15 cfm.

Internally lubricated compressors may produce carbon monoxide upon overheating, and it is advisable to have a continuous carbon monoxide monitor or a compressor overheating alarm with an automatic shutdown device to safeguard the system. Care must be given to the location of the compressor air intake to

prevent contamination of the breathing air by the operation of vehicles or other equipment that generate toxic fumes close to the intake.

A chemical cartridge respirator equipped with an organic vapour cartridge will filter isocyanates effectively. The problem with its use is that isocyanates have a relatively high odour threshold (0.4 ppm for TDI) in comparison to the TWAE; therefore, the wearer might be exposed to a high concentration before being warned that the cartridges are saturated and no longer effective.

The organic vapour cartridge respirator does afford protection against many paint solvents, and, when fitted with an additional filter, will protect against paint and enamel mists containing metallic pigments. It is therefore acceptable as protection against paints that do not contain isocyanates. Until recently, painters either rejected respiratory protection completely, or else used a mechanical filter mask consisting of a few layers of gauze mounted in an aluminum frame. This respirator provides little protection against any of the hazards and should not be used under *any* circumstances. Since the use of a supplied-air respirator is mandatory for isocyanate-containing paints, it is suggested that it be used for all spray painting within the spray booth.

A mechanical filter respirator that provides protection against toxic dusts should be worn by bodymen when sanding body putty. A number of disposable dust respirators are now available that provide that protection. Many companies are now providing this type of respirator to their employees.

Finally, a supplied-air respirator is also required for protection against the inhalation of dust during abrasive blasting. This type of respiratory protection consists of an abrasion-resistant hood, to which compressed air is supplied in a continuous flow to maintain the hood under positive pressure.

The use of toxic dust filter respirators is not considered acceptable protection due to the extremely high dust concentrations generated by abrasive blasting.

For all of these respirator applications, consideration must be given to organizing a respirator program that should include provisions for worker training and adequate respirator cleaning, and that takes into consideration all the general requirements listed in the Code for Respiratory Equipment for Isocyanates.

Skin and Eye Protection

Protective clothing, including coveralls and impervious gloves, should be worn whenever the risk of skin contact with paints and solvents exists. Safety goggles or a facemask should be worn to prevent eye injury from splashes. In addition, an eye-wash station should be provided to allow prompt irrigation of the eyes, should such an accident occur. Strict personal hygiene should be promoted, including thorough washing before meals and at the end of the workday. Solvents should never be used to clean paint from the skin; rather a commercial hand cleaner should be used.

Hearing Protection

To prevent noise-induced hearing loss caused by exposure to pneumatic chisels, sanders and welders, hearing protection should be worn by workers while using these tools. There are a variety of ear plugs and muffs available which, if worn correctly, provide sufficient noise attentuation to reduce the worker noise exposure to an acceptable level.

Recommendations

In light of the general acceptance of isocyanate-containing paints, a sound industrial hygiene control program for the automotive body refinishing industry should include the following components:

1. The use of a well-ventilated spray booth to isolate the paint fumes from the general shop area.

2. The use of supplied-air respiratory protection by the spray painter inside the spray booth.

3. The use of a toxic dust filter respirator by bodymen when sanding body putty.

4. The use of a hood-type supplied-air respirator while abrasive blasting.

5. The provision of clean, high-quality compressed breathing air to supplied-air respirators.

6. The wearing of protective equipment to prevent skin and eye injury due to contact with paints and solvents.

7. The use of hearing protection by workers exposed to high noise levels generated by pneumatic tools.

Appendix

Code for Respiratory Equipment for Isocyanates (Ont. Reg. 455/83)

Type of Respirator Required

The respiratory equipment provided by an employer and used by a worker shall meet or exceed the following requirements:

Airborne Concentration (Isocyanate vapour and particulates)	*Type of Respirator Required*
Less than or equal to 0.25 ppm or 10 µmol/m³ (50 X TWAE)	Positive pressure supplied air respirator with full face-piece or hood or self-contained breathing apparatus with full face-piece.
Greater than 0.25 ppm or 10 µmol/m³ (50 X TWAE)	A NIOSH Type C supplied air respirator operated in pressure demand or other positive pressure or continuous flow mode or self-contained breathing apparatus with a full face-piece operated in pressure demand.
Escape	Any escape self-contained breathing apparatus with full face-piece operated in pressure demand.

Notes:
1. *Vapours* can be defined as the gaseous form of a substance which is normally either a liquid or a solid at ordinary temperatures and pressures.
2. Respirators need not be worn if the levels of isocyanates in air are less than the TWAE of 0.005 ppm. However, if the worker wishes to use a respirator below the TWAE, the organic vapour/particulate air purifying respirator (NIOSH/MESA T23C) can be used.
3. Supplied air respirator does not include a powered air purifying respirator.

General Requirements For the Use of Respirators

1. Subject to the Table in this Code, respiratory protective equipment must be used in accordance with the procedure specified by the equipment manufacturer.

2. Written standard operating procedures governing the selection and use of respirators must be established.

3. Written procedures on the use and care of respirators must be reviewed with workers.

4. Workers must be trained in the proper use of respirators and their limitations.

5. Respirators must be fitted so that there is an effective seal between the respirator and the worker's face.

6. Where practicable, the respirators are to be assigned to individual workers for their exclusive use.

7. Compressed air, compressed oxygen, liquid air and liquid oxygen used for respirators are to meet CSA standards when applicable.

8. Respirators must be regularly cleaned, disinfected and inspected. Those issued for the exclusive use of one worker must be cleaned after each day's use, or more often if necessary, while those used by more than one worker must be thoroughly cleaned and disinfected after each use.

9. Worn or deteriorated parts must be replaced.

10. Respirators for emergency use, such as self-contained devices, are to be thoroughly tested at least once a month, and serviced after each use.

11. All respirators and replacement parts must be stored in a convenient, clean and sanitary location.

12. Persons must not be assigned to tasks requiring use of respirators unless they are physically able to perform the work and use the equipment.

13. Workers required to wear respirators who experience breathing difficulty while using

respirators must be referred to a physician for evaluation.

14. Records of frequency of use by the worker of respiratory equipment and its type are to be kept.

Résumé

Les résultats d'enquêtes sur l'hygiène en milieu industriel figurent ci-après. Elles one été menées dans cinq ateliers de débosselage pour déterminer le degré de danger associé à l'utilisation de peintures et de durcisseurs contenant des isocyanates.

On a utilisé un appareillage de prélèvement d'échantillons formé de deux minuscules conimètres à impact en série, contenant chacun une solution absorbante réactive à l'azote. L'analyse s'est faite par chromatographie liquide à grande vitesse. Les résultats varient de moins de 0,001 à 0,003 ppm. Cependant, les composés d'isocyanate présents dans les peintures sont sous forme prépolymérisée, et l'exposition combinée aux formes monomères et polymères peut excéder 0,005 ppm.

Des suggestions ont été faites concernant la ventilation par aspiration et la protection du peintre de façon à ce qu'il soit le moins possible exposé à la peinture appliquée au pistolet.

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Guideline for Examining Physicians on Surveillance of Silica-Exposed Workers

Purpose of Medical Surveillance

1. To detect early, asymptomatic silica-related disease.
2. To identify individuals at increased risk of developing respiratory impairment if silicosis were to develop.
3. To prevent progression to total disability.

Risk Group

The inhalation of crystalline silica dust on a prolonged or intense basis is a hazard to health. At special risk are workers already impaired by respiratory disease and those with a history of inadequately treated tuberculosis.

Known Respiratory Sequelae

The silica-related respiratory diseases are: nodular silicosis, mixed dust fibrosis, alveolar lipoproteinosis with diffuse interstitial pulmonary fibrosis, conglomerate silicosis and silicotuberculosis. Certain syndromes are found with increased frequency in association with silica exposure, but are not specific to it: scar or irregular emphysema, compensatory emphysema, cor pulmonale, spontaneous pneumothorax, segmental or middle lobe atelectasis, rheumatoid syndrome, scleroderma, left recurrent laryngeal nerve paralysis from lymph node compression and oesophageal compression from lymph nodes.

Pre-Placement Examination

1. Medical history, with emphasis on past occupational exposure, smoking history and past or current respiratory problems, including tuberculosis.
2. Physical examination with special attention to the respiratory system.
3. Chest postero-anterior X-ray with optional lateral view.
4. Forced expiratory spirometry done to American Thoracic Society standards.

Pre-Employment Risk Factors	Inadequately treated tuberculosis and any currently active significant respiratory disease are contra-indications to employment in silica exposure. Healed respiratory illness that has not led to a moderate ventilatory defect on spirometry or major disorganization of the pulmonary architecture is not necessarily a contra-indication to employment. Under investigation, but not established as risk factors in humans, are the presence of circulating antinuclear antibodies, rheumatoid disease and recurrent non-tuberculous chest infections.
Screening During Employment	Chest PA X-ray and forced expiratory spirometry at least every second year.
Action Levels	**Radiographic** The commonest initial presentation of simple silicosis is the finding of small rounded opacities in the upper zones of the lungs. Before the diagnosis becomes certain, the appearance may be similar to the ILO 80 category 1/0. The suspicion of possible radiographic pneumoconiosis is recorded as Ontario Code '4'. The definite radiographic diagnosis is coded '5'. Ultimately, silicosis is diagnosed on the combination of the characteristic X-ray appearance (at least ILO 1/1), adequacy of exposure to free silica dust, absence of identifiable confounding disease and knowledge of an incidence of clustering of similar cases. It is recognized that very intense exposures, such as are met in sandblasting, may lead to typical and accelerated presentations. **Physiologic** Spirometry will not be abnormal before the stage of conglomerate silicosis. Any earlier ventilatory defects indicate intercurrent disease, which may pose a risk of impairment, but should not be interpreted as silica-related in the absence of radiographic evidence. A moderate ventilatory defect is significant, whereas slight defects may overlap with the normal range and should be interpreted in the light of additional evidence for disease. **Pathologic** While carefully reviewed pathologic material may be helpful in the handling of some difficult claims, the diagnosis is successfully made on the basis of clinical findings and non-invasive studies in the vast majority of cases.

Management of Suspected Abnormals

The following *pre-employment* findings are relative contra-indications to permanent employment in silica exposure:

1. Any currently active respiratory disease.

2. Inadequately treated tuberculosis.

3. A respiratory illness that has caused a moderate ventilatory defect or major disorganization of the pulmonary architecture.

Isolated abnormalities, especially on spirometry, should be reviewed for quality of effort and repeated if necessary. Temporary airflow limitation thought to be due to hyper-reactive airways syndrome (usually asymptomatic), should be reassessed after a suitable interval. Consultation with the medical staff of the Chest Clinic of the Occupational Health Branch is recommended in borderline cases or contentious situations.

Abnormalities found *during employment* will require repeat testing if films or spirometry are of questionable quality, clinical examination and, possibly, local referral to a chest specialist to consider the presence of confounding disease, and consultation with medical staff of the Chest Clinic of the Occupational Health Branch.

A recommendation of *removal from work* because of occupational respiratory disease shall be made only after joint review with the senior medical consultant of the Ministry of Labour, Occupational Health Branch Chest Clinic and with the chest consultant to the Workers' Compensation Board.

A claim with the WCB should be initiated if silicosis is thought to be present. It should be appreciated by affected workers that early silicosis does not, in itself, lead to functional impairment, and a claim may not be allowed as long as the silicosis remains functionally slight.

Publications Available From the Government Book Store

Publications under the Regulation Act vol. 112-39	
Revised Regulations of Ontario, 1980 Regulation 692/80 Industrial Establishments	$2.00
Revised Regulations of Ontario, 1980 Regulation 691/80 Construction Projects	$2.00
Revised Regulations of Ontario, 1980 Regulation 694/80 Mines and Mining Plants	$2.00
Regulation respecting Lead - made under the Occupational Health and Safety Act Ontario Regulation 536/81	$1.00
Regulation respecting Mercury - made under the Occupational Health and Safety Act Ontario Regulation 141/82	$1.00
Regulation Respecting Vinyl Chloride - made under the Occupational Health and Safety Act Ontario Regulation 516/82	$1.00
Regulation respecting Coke Oven Emissions - made under the Occupational Health and Safety Act Ontario Regulation 517/82	$1.00
Regulation respecting Asbestos - made under the Occupational Health and Safety Act Ontario Regulation 570/82	$1.00
Regulation respecting Isocyanates - made under the Occupational Health and Safety Act Ontario Regulation 455/83	$1.00

An Occupational Health Nurse's Guide to the Preparation of a Policy and Procedure Manual	$2.00
A Guide to the Occupational Health and Safety Act	$1.00
Regulation for X-ray Safety, Registration and Plan Review made under the Occupational Health and Safety Act Ontario Regulation 263/84	.60
X-ray Safety Code, Ontario Regulation 45/84	.20

Coming Events

October 15-17, 1984	International Conference on Organic Solvent Toxicity. Stockholm, Sweden.
October 14-18, 1984	International Conference on Occupational Radiation Safety in Mining. Toronto, Ontario, Canada. Details from Canadian Nuclear Association, 111 Elizabeth Street, 11th Floor, Toronto, Ontario, Canada, M5G 1P7.
October 29-November 2, 1984	1984 Ontario Occupational Health Nurses Association Conference. Cleary Convention Centre, Windsor, Ontario, Canada. For further information contact Lorraine Carnelos, Reg. N., ECOHNA, 1600 Lauzon Road, Windsor, Ontario, Canada, N9A 6R9.
October 30-November 1, 1984	Conference on Protective Equipment. Constellation Hotel, Toronto. Details from Canadian Standards Association, (COPE '84) 178 Rexdale Blvd., Rexdale, Ontario, Canada, M9W 1R3.
November 28-30, 1984	Occupational Medical Association of Canada, 2nd Annual Scientific Meeting. Bonaventure Hotel, Montreal. For further information contact Dr. Michel Joncas, 1028 Chemin du Lac Levy, Longueil, P.Q., Canada, J6N 1A8.
December 3-5, 1984	Inter-Noise 84 - 13th International Conference on Noise Control Engineering - Honolulu. Details from the Inter-Noise 84 Secretariat, P.O. Box 3649, Arlington Branch, Poughkeepsie, NY 12603, U.S.A.
December 7-8, 1984	2nd Annual Recent Advances in Occupational Cancer. San Francisco, CA. Information: Extended Programs in Medical Education, Rm 569-U, University of California, San Francisco, CA 94143; 415/666-4251.
October 1-3, 1985	Ventilation '85, 1st International Symposium on Ventilation for Contaminant Control. Royal York Hotel, Toronto, Canada. For details contact Dr. H.D. Goodfellow, Hatch Associates Ltd., 21 St. Clair Ave. East, Toronto, Ontario, Canada, M4T 1L9.

Ontario Ministry of Labour

Occupational Health and Safety Division

Occupational Health Branch

400 University Ave.
Toronto, Ontario
M7A 1T7

Telephone:
(416) 965-3610

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